

LEWIS 10/ 537 704 = chi conotoxins (Group I; Clms 1-7; SEQ ID NO: 4)

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NEWS 5 AUG 30 CA(SM)/CAplus(SM) Austrian patent law changes
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NEWS 10 SEP 25 CAS REGISTRY(SM) updated with amino acid codes for
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NEWS 14 OCT 23 Option to turn off MARPAT highlighting enhancements available
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NEWS 21 NOV 20 CAS Registry Number crossover limit increased to 300,000 in
additional databases
NEWS 22 NOV 20 CA/CAplus to MARPAT accession number crossover limit
increased
to 50,000
NEWS 23 DEC 01 CAS REGISTRY updated with new ambiguity codes
NEWS 24 DEC 11 CAS REGISTRY chemical nomenclature enhanced
NEWS 25 DEC 14 WPIDS/WPINDEX/WPIX manual codes updated
NEWS 26 DEC 14 GBFULL and FRFULL enhanced with IPC 8 features and
functionality
NEWS 27 DEC 18 CA/CAplus pre-1967 chemical substance index entries enhanced
with preparation role
NEWS 28 DEC 18 CA/CAplus patent kind codes updated
NEWS 29 DEC 18 MARPAT to CA/CAplus accession number crossover limit
increased
to 50,000
NEWS 30 DEC 18 MEDLINE updated in preparation for 2007 reload
NEWS EXPRESS NOVEMBER 10 CURRENT WINDOWS VERSION IS V8.01c, CURRENT

MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 25 SEPTEMBER 2006.

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FILE 'HOME' ENTERED AT 17:36:32 ON 21 DEC 2006

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DICTIONARY FILE UPDATES: 20 DEC 2006 HIGHEST RN 916134-56-0

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=> s XGVCCGYKLCHXC/sqsp
L1 11 XGVCCGYKLCHXC/SQSP

=> D CN SQL SEQ 1-11

L1 ANSWER 1 OF 11 REGISTRY COPYRIGHT 2006 ACS on STN
CN Peptide, (Xaa-Gly-Val-Cys-Cys-Gly-Tyr-Lys-Leu-Cys-His-Xaa-Cys) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 5: PN: WO2004050690 SEQID: 4 unclaimed protein
SQL 13

SEQ 1 XGVCCGYKLC HXC
===== ===
HITS AT: 1-13

L1 ANSWER 2 OF 11 REGISTRY COPYRIGHT 2006 ACS on STN
CN L-Cysteine, N5-(aminocarbonyl)-L-ornithylglycyl-L-valyl-L-cysteinyl-L-cysteinylglycyl-L-tyrosyl-L-lysyl-L-leucyl-L-cysteinyl-L-histidyl-(4R)-4-hydroxy-L-prolyl-, cyclic (4.fwdarw.13), (5.fwdarw.10)-bis(disulfide) (9CI) (CA INDEX NAME)

OTHER NAMES:
CN 128: PN: WO2004050688 SEQID: 139 claimed protein
SQL 13

SEQ 1 XGVCCGYKLC HXC
===== ===
HITS AT: 1-13

L1 ANSWER 3 OF 11 REGISTRY COPYRIGHT 2006 ACS on STN
CN L-Cysteine, L-norleucylglycyl-L-valyl-L-cysteinyl-L-cysteinylglycyl-L-tyrosyl-L-lysyl-L-leucyl-L-cysteinyl-L-histidyl-(4R)-4-hydroxy-L-prolyl-, cyclic (4.fwdarw.13), (5.fwdarw.10)-bis(disulfide) (9CI) (CA INDEX NAME)

OTHER NAMES:
CN 108: PN: WO2004050688 SEQID: 119 claimed protein
SQL 13

SEQ 1 XGVCCGYKLC HXC
===== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

L1 ANSWER 4 OF 11 REGISTRY COPYRIGHT 2006 ACS on STN
CN L-Cysteine, N3-(N-acetyl-L-tryptophyl)-(3S)-3,7-diaminoheptanoylglycyl-L-valyl-L-cysteinyl-L-cysteinylglycyl-L-tyrosyl-L-lysyl-L-leucyl-L-cysteinyl-L-histidyl-(4R)-4-hydroxy-L-prolyl-, cyclic (5.fwdarw.14), (6.fwdarw.11)-bis(disulfide) (9CI) (CA INDEX NAME)

OTHER NAMES:
CN 98: PN: WO2004050688 SEQID: 109 claimed sequence
SQL 14

SEQ 1 WXGVCCGYKL CHXC
===== ===
HITS AT: 2-14

L1 ANSWER 5 OF 11 REGISTRY COPYRIGHT 2006 ACS on STN
CN L-Cysteine, L-norleucylglycyl-L-valyl-L-cysteinyl-L-cysteinylglycyl-O-methyl-L-tyrosyl-L-lysyl-L-leucyl-L-cysteinyl-L-histidyl-(4R)-4-hydroxy-L-prolyl-, cyclic (4.fwdarw.13), (5.fwdarw.10)-bis(disulfide) (9CI) (CA INDEX NAME)

OTHER NAMES:
CN 96: PN: WO2004050688 SEQID: 107 claimed protein
SQL 13

SEQ 1 XGVCCGYKLC HXC
===== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

L1 ANSWER 6 OF 11 REGISTRY COPYRIGHT 2006 ACS on STN
CN L-Cysteinamide, 5-oxo-D-prolylglycyl-L-valyl-L-cysteinyl-L-cysteinylglycyl-

L-tyrosyl-L-lysyl-L-leucyl-L-cysteinyl-L-histidyl-(4R)-4-hydroxy-L-prolyl-, cyclic (4.fwdarw.13), (5.fwdarw.10)-bis(disulfide) (9CI) (CA INDEX NAME)
SQL 13

SEQ 1 XGVCCGYKLC HXC
===== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

L1 ANSWER 7 OF 11 REGISTRY COPYRIGHT 2006 ACS on STN
CN L-Cysteine, 5-oxo-D-prolylglycyl-L-valyl-L-cysteinyl-L-cysteinylglycyl-L-tyrosyl-L-lysyl-L-leucyl-L-cysteinyl-L-histidyl-(4R)-4-hydroxy-L-prolyl-, cyclic (4.fwdarw.13), (5.fwdarw.10)-bis(disulfide) (9CI) (CA INDEX NAME)

OTHER NAMES:

CN 133: PN: WO2004050688 SEQID: 144 claimed protein
SQL 13

SEQ 1 XGVCCGYKLC HXC
===== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

L1 ANSWER 8 OF 11 REGISTRY COPYRIGHT 2006 ACS on STN
CN L-Cysteine, 5-oxo-L-prolylglycyl-L-valyl-L-cysteinyl-L-cysteinylglycyl-L-tyrosyl-L-lysyl-L-leucyl-L-cysteinyl-L-histidyl-(4R)-4-hydroxy-L-prolyl-, cyclic (4.fwdarw.13), (5.fwdarw.10)-bis(disulfide) (9CI) (CA INDEX NAME)

OTHER NAMES:

CN 131: PN: WO2004050688 SEQID: 142 claimed protein
SQL 13

SEQ 1 XGVCCGYKLC HXC
===== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

L1 ANSWER 9 OF 11 REGISTRY COPYRIGHT 2006 ACS on STN
CN L-Cysteinamide, 5-oxo-L-prolylglycyl-L-valyl-L-cysteinyl-L-cysteinylglycyl-O-methyl-L-tyrosyl-L-lysyl-L-leucyl-L-cysteinyl-L-histidyl-(4R)-4-hydroxy-L-prolyl-, cyclic (4.fwdarw.13), (5.fwdarw.10)-bis(disulfide) (9CI) (CA INDEX NAME)

SQL 13

SEQ 1 XGVCCGYKLC HXC
===== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

L1 ANSWER 10 OF 11 REGISTRY COPYRIGHT 2006 ACS on STN
CN D-Cysteinamide, 5-oxo-L-prolylglycyl-L-valyl-L-cysteinyl-L-cysteinylglycyl-L-tyrosyl-L-lysyl-L-leucyl-L-cysteinyl-L-histidyl-(4R)-4-hydroxy-L-prolyl-, cyclic (5.fwdarw.10)-disulfide (9CI) (CA INDEX NAME)

SQL 13

SEQ 1 XGVCCGYKLC HXC
===== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

L1 ANSWER 11 OF 11 REGISTRY COPYRIGHT 2006 ACS on STN
CN L-Cysteinamide, 5-oxo-L-prolylglycyl-L-valyl-L-cysteiny-L-
cysteinyglycyl-
L-tyrosyl-L-lysyl-L-leucyl-L-cysteiny-L-histidyl-(4R)-4-hydroxy-L-prolyl-
, cyclic (4.fwdarw.13), (5.fwdarw.10)-bis(disulfide) (9CI) (CA INDEX NAME)

SQL 13

SEQ 1 XGVCCGYKLC HXC

=====

HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

=> file CAPLUS

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

99.73

99.94

FULL ESTIMATED COST

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=> s L1 and PATENT/DT

2 L1

5541564. PATENT/DT

L2 2 L1 AND PATENT/DT

=> D L2 BIB ABS

L2 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2006 ACS on STN

AN 2004:493731 CAPLUS

DN 141:47381

TI Therapeutic .chi.-conotoxin peptides (-I)

IN Lewis, Richard James; Alewood, Paul Francis; Alewood, Dianne; Palant, Elka

PA Xenome Ltd., Australia

SO PCT Int. Appl., 57 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004050690	A1	20040617	WO 2003-AU1605	20031202
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2508129	A1	20040617	CA 2003-2508129	20031202
AU 2003302609	A1	20040623	AU 2003-302609	20031202
EP 1572725	A1	20050914	EP 2003-812100	20031202
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2006523181	T	20061012	JP 2004-555869	20031202
US 2006142201	A1	20060629	US 2005-537704	20051212
PRAI US 2002-430306P	P	20021202		
WO 2003-AU1605	W	20031202		

instant application

OS MARPAT 141:47381

AB The invention discloses an isolated, synthetic or recombinant .chi.-conotoxin peptide comprising the sequence Xaa1-Xaa2-Gly-Val-Cys-Cys-Gly-Tyr-Lys-Leu-Cys-His-Pro-Cys (Xaa1 = N-terminal pyroglutamate, D-pyroglutamate; Xaa2 = Asn, deletion; or such a sequence in which .gtoreq.1 Cys is replaced with corresponding D-amino acid and/or one or more amino acid residues other than Cys has undergone a side chain modification), or a salt, ester, amide or prodrug thereof. The invention also discloses pharmaceutical compns. comprising these peptides, as well as the use of these peptides in the prophylaxis or treatment of conditions, such as, but not limited to, pain, inflammation, incontinence, cardiovascular conditions, and mood disorders.

RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d L2 1 BIB ABS

L2 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2004:493731 CAPLUS
DN 141:47381
TI Therapeutic .chi.-conotoxin peptides (-I)
IN Lewis, Richard James; Alewood, Paul Francis; Alewood, Dianne; Palant, Elka
PA Xenome Ltd., Australia
SO PCT Int. Appl., 57 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004050690	A1	20040617	WO 2003-AU1605	20031202
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK,				

TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

CA 2508129	A1	20040617	CA 2003-2508129	20031202
AU 2003302609	A1	20040623	AU 2003-302609	20031202
EP 1572725	A1	20050914	EP 2003-812100	20031202

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK

JP 2006523181	T	20061012	JP 2004-555869	20031202
US 2006142201	A1	20060629	US 2005-537704	20051212

PRAI US 2002-430306P P 20021202

WO 2003-AU1605 W 20031202

OS MARPAT 141:47381

AB The invention discloses an isolated, synthetic or recombinant .chi.-conotoxin peptide comprising the sequence Xaa1-Xaa2-Gly-Val-Cys-Cys-Gly-Tyr-Lys-Leu-Cys-His-Pro-Cys (Xaa1 = N-terminal pyroglutamate, D-pyroglutamate; Xaa2 = Asn, deletion; or such a sequence in which .gtoreq.1 Cys is replaced with corresponding D-amino acid and/or one or more amino acid residues other than Cys has undergone a side chain modification), or a salt, ester, amide or prodrug thereof. The invention also discloses pharmaceutical compns. comprising these peptides, as well as the use of these peptides in the prophylaxis or treatment of conditions, such as, but not limited to, pain, inflammation, incontinence, cardiovascular conditions, and mood disorders.

RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d L2 2 BIB ABS

L2 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2006 ACS on STN

AN 2004:493729 CAPLUS

DN 141:47357

TI Neuronal amine transporter-inhibiting .chi.-conotoxin peptides (-II)

IN Lewis, Richard James; Alewood, Paul Francis; Alewood, Dianne; Palant, Elka

PA Xenome Ltd., Australia

SO PCT Int. Appl., 156 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI	WO 2004050688	A1	20040617	WO 2003-AU1606	20031202	
	W:			AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW		
	RW:			BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG		
	AU 2003302610	A1	20040623	AU 2003-302610	20031202	
	EP 1578787	A1	20050928	EP 2003-812101	20031202	
	R:			AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK		
	US 2006270832	A1	20061130	US 2005-537088	20051227	
PRAI	US 2002-430307P	P	20021202			
	WO 2003-AU1606	W	20031202			

OS MARPAT 141:47357

AB An isolated, synthetic or recombinant .chi.-conotoxin peptide having the ability to inhibit neuronal amine transporter comprises Cys-Cys-Gly-Tyr-Lys-Leu-Cys-Xaa5-Xaa6-Cys (Xaa5, Xaa6 = absent or any

amino acid residue except Cys, or a sequence in which Gly, Tyr, Lys or Leu are subject to conservative amino acid substitution or side chain modification; with proviso), or a salt, ester, amide, prodrug or cyclized deriv. thereof.

RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> file biosis embase medline agricola
COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
12.01	111.95

FULL ESTIMATED COST

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE	TOTAL
ENTRY	SESSION
-2.25	-2.25

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FILE 'AGRICOLA' ENTERED AT 17:40:51 ON 21 DEC 2006

=> s L1
L3 0 L1

=> s chi conotoxin
L4 12 CHI CONOTOXIN

=> d L4 1-12 BIB ABS

L4 ANSWER 1 OF 12 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
AN 2006:645200 BIOSIS
DN PREV200600636757
TI Cyclic MrIA: A stable and potent cyclic conotoxin with a novel topological fold that targets the norepinephrine transporter.
AU Lovelace, Erica S.; Armishaw, Christopher J.; Colgrave, Michelle L.; Wahlstrom, Maria E.; Alewood, Paul F.; Daly, Norelle L.; Craik, David J. [Reprint Author]
CS Univ Queensland, Inst Mol Biosci, Brisbane, Qld 4072, Australia
d.craik@imb.uq.edu.au
SO Journal of Medicinal Chemistry, (NOV 2 2006) Vol. 49, No. 22, pp. 6561-6568.
CODEN: JMCMAR. ISSN: 0022-2623.
DT Article
LA English
ED Entered STN: 22 Nov 2006
Last Updated on STN: 22 Nov 2006
AB Conotoxins, disulfide-rich peptides from the venom of cone snails, have created much excitement over recent years due to their potency and specificity for ion channels and their therapeutic potential. One recently identified conotoxin, MrIA, a 13-residue member of the chi-conotoxin family, inhibits the human norepinephrine transporter (NET) and has potential applications in the treatment of pain. In the current study, we show that the beta-hairpin structure of native MrIA is retained in a synthetic cyclic version, as is biological activity at the NET. Furthermore, the cyclic version has increased resistance to trypsin digestion relative to the native peptide, an intriguing result because the cleavage site for the trypsin is not close to the cyclization

site. The use of peptides as drugs is generally hampered by susceptibility to proteolysis, and so, the increase in enzymatic stability against trypsin observed in the current study may be useful in improving the therapeutic potential of MrIA. Furthermore, the structure reported here for cyclic MrIA represents a new topology among a growing number of circular disulfide-rich peptides.

L4 ANSWER 2 OF 12 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
AN 2006:30215 BIOSIS
DN PREV200600030783
TI Backbone cyclization improves the enzymatic stability of chiconotoxin, MrIA, whilst maintaining its structure and NET-modulating activity.
AU Lovelace, Erica S. [Reprint Author]; Armishaw, Christopher J.; Colgrave, Michelle L.; Alewood, Paul F.; Daly, Norelle L.; Craik, David J.
CS Univ Queensland, Inst Mol Biosci, Brisbane, Qld 4072, Australia
SO Biopolymers, (2005) Vol. 80, No. 4, pp. 585.
Meeting Info.: 19th American Peptide Symposium. San Diego, CA, USA. June 18 -23, 2005. Amer Peptide Soc; AAPPTEC; Amer Peptide Co; Amer Hlth/GE Healthcare; Amgen Inc; BACHEM; BIOMOL Int; C S Bio Co; Cambridge Res Biochem; Chemico Int Inc; Chem Today; Eli Lilly & Co; ESCOM Sci Fdn; Genentech; Hoffman-La Roche Inc; Merck Res Lab; Midwest Bio-Tech Inc; NeomPS Inc; New England Biolabs Inc; Novo Nordisk A/S; Peptides Int Inc; PharmaChem; PolyPeptide Lab Inc; RSP Amino Acide LLC; Senn Chem USA; Sinopep Pharmaceut Inc; SynPep Corp; Synthetech Inc; UCB Bioproducts Inc. CODEN: BIPMAA. ISSN: 0006-3525.
DT Conference; (Meeting)
Conference; (Meeting Poster)
LA English
ED Entered STN: 28 Dec 2005
Last Updated on STN: 28 Dec 2005

L4 ANSWER 3 OF 12 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
AN 2004:422061 BIOSIS
DN PREV200400427664
TI Peptides.
AU Lewis, Richard James [Inventor, Reprint Author]; Alewood, Paul Francis [Inventor]; Sharpe, Iain Andrew [Inventor]
CS Woolloongabba, Australia
ASSIGNEE: The University of Queensland, Queensland, Australia
PI US 6794361 20040921
SO Official Gazette of the United States Patent and Trademark Office Patents, (Sep 21 2004) Vol. 1286, No. 3. <http://www.uspto.gov/web/menu/patdata.html>. e-file.
ISSN: 0098-1133 (ISSN print).
DT Patent
LA English
ED Entered STN: 3 Nov 2004
Last Updated on STN: 3 Nov 2004
AB The invention relates to an isolated, synthetic or recombinant chiconotoxin peptide having the ability to inhibit a neuronal amine transporter, nucleic acid molecules encoding all or part of such peptides, antibodies to such peptides and uses and methods of treatment involving them.

L4 ANSWER 4 OF 12 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
AN 2006557072 EMBASE
TI Therapeutic applications of conotoxins that target the neuronal nicotinic acetylcholine receptor.
AU Livett B.G.; Sandall D.W.; Keays D.; Down J.; Gayler K.R.; Satkunanathan N.; Khalil Z.
CS B.G. Livett, Department of Biochemistry and Molecular Biology, Bio21

Molecular Science and Biotechnology Institute, University of Melbourne,
 Vic. 3010, Australia. b.livett@unimelb.edu.au

SO Toxicon, (1 Dec 2006) Vol. 48, No. 7, pp. 810-829. .
 Refs: 130
 ISSN: 0041-0101 CODEN: TOXIA6

PUI S 0041-0101(06)00249-2

CY United Kingdom

DT Journal; Article

FS 008 Neurology and Neurosurgery
 030 Pharmacology
 037 Drug Literature Index
 038 Adverse Reactions Titles
 052 Toxicology

LA English

SL English

ED Entered STN: 1 Dec 2006
 Last Updated on STN: 1 Dec 2006

AB Pain therapeutics discovered by molecular mining of the expressed genome
 of Australian predatory cone snails are providing lead compounds for the
 treatment of neurological diseases such as multiple sclerosis, shingles,
 diabetic neuropathy and other painful neurological conditions. The high
 specificity exhibited by these novel compounds for neuronal receptors and
 ion channels in the brain and nervous system indicates the high degree of
 selectivity that this class of neuropeptides can be expected to show when
 used therapeutically in humans. A lead compound, ACV1 (conotoxin Vc1.1
 from *Conus victoriae*), has entered Phase II clinical trials and is being
 developed for the treatment for neuropathic pain. ACV1 will be targeted
 initially for the treatment of sciatica, shingles and diabetic neuropathy.
 The compound is a 16 amino acid peptide [Sandall et al., 2003. A novel
 .alpha.-conotoxin identified by gene sequencing is active in suppressing
 the vascular response to selective stimulation of sensory nerves in vivo.
 Biochemistry 42, 6904-6911], an antagonist of neuronal nicotinic
 acetylcholine receptors. It has potent analgesic activity following
 subcutaneous or intramuscular administration in several preclinical animal
 models of human neuropathic pain [Satkunanathan et al., 2005. Alpha
 conotoxin Vc1.1 alleviates neuropathic pain and accelerates functional
 recovery of injured neurons. Brain. Res. 1059, 149-158]. ACV1 may act
 as an analgesic by decreasing ectopic excitation in sensory nerves. In
 addition ACV1 appears to accelerate the recovery of injured nerves and
 tissues. .COPYRGT. 2006.

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AN 2006532716 EMBASE

TI Cyclic MrIA: A stable and potent cyclic conotoxin with a novel topological
 fold that targets the norepinephrine transporter.

AU Lovelace E.S.; Armishaw C.J.; Colgrave M.L.; Wahlstrom M.E.; Alewood P.F.;
 Daly N.E.; Craik D.J.

CS D.J. Craik, Institute for Molecular Bioscience, University of Queensland,
 Brisbane, QLD 4072, Australia. d.craik@imb.uq.edu.au

SO Journal of Medicinal Chemistry, (2 Nov 2006) Vol. 49, No. 22, pp.
 6561-6568. .
 Refs: 59
 ISSN: 0022-2623 CODEN: JMCMAR

CY United States

DT Journal; Article

FS 008 Neurology and Neurosurgery
 029 Clinical Biochemistry
 030 Pharmacology
 037 Drug Literature Index

LA English

SL English

ED Entered STN: 24 Nov 2006

Last Updated on STN: 24 Nov 2006

AB Conotoxins, disulfide-rich peptides from the venom of cone snails, have created much excitement over recent years due to their potency and specificity for ion channels and their therapeutic potential. One recently identified conotoxin, MrIA, a 13-residue member of the .chi.-conotoxin family, inhibits the human norepinephrine transporter (NET) and has potential applications in the treatment of pain. In the current study, we show that the .beta.-hairpin structure of native MrIA is retained in a synthetic cyclic version, as is biological activity at the NET. Furthermore, the cyclic version has increased resistance to trypsin digestion relative to the native peptide, an intriguing result because the cleavage site for the trypsin is not close to the cyclization site. The use of peptides as drugs is generally hampered by susceptibility to proteolysis, and so, the increase in enzymatic stability against trypsin observed in the current study may be useful in improving the therapeutic potential of MrIA. Furthermore, the structure reported here for cyclic MrIA represents a new topology among a growing number of circular disulfide-rich peptides. .COPYRGT. 2006 American Chemical Society.

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AN 2006281221 EMBASE

TI Synthetic .mu.O-conotoxin MrVIB blocks TTX-resistant sodium channel Na (V)1.8 and has a long-lasting analgesic activity.

AU Bulaj G.; Zhang M.-M.; Green B.R.; Fiedler B.; Layer R.T.; Wei S.; Nielsen J.S.; Low S.J.; Klein B.D.; Wagstaff J.D.; Chicoine L.; Harty T.P.; Terlau H.; Yoshikami D.; Olivera B.M.

CS G. Bulaj, Department of Biology, University of Utah, 257 S. 1400 East, Salt Lake City, UT 84112, United States. bulaj@biology.utah.edu

SO Biochemistry, (13 Jun 2006) Vol. 45, No. 23, pp. 7404-7414. .

Refs: 47

ISSN: 0006-2960 CODEN: BICHAW

CY United States

DT Journal; Article

FS 024 Anesthesiology
029 Clinical Biochemistry
037 Drug Literature Index
052 Toxicology

LA English

SL English

ED Entered STN: 30 Jun 2006

Last Updated on STN: 30 Jun 2006

AB .mu.O-Conotoxin MrVIB is a blocker of voltage-gated sodium channels, including TTX-sensitive and -resistant subtypes. A comprehensive characterization of this peptide has been hampered by the lack of sufficient synthetic material. Here, we describe the successful chemical synthesis and oxidative folding of MrVIB that has made an investigation of the pharmacological properties and therapeutic potential of the peptide feasible. We show for the first time that synthetic MrVIB blocks rat Na(V)1.8 sodium channels and has potent and long-lasting local anesthetic effects when tested in two pain assays in rats. Furthermore, MrVIB can block propagation of action potentials in A- and C-fibers in sciatic nerve as well as skeletal muscle in isolated preparations from rat. Our work provides the first example of analgesia produced by a conotoxin that blocks sodium channels. The emerging diversity of antinociceptive mechanisms targeted by different classes of conotoxins is discussed. .COPYRGT. 2006 American Chemical Society.

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AN 2005185483 EMBASE

TI Molecular prospecting for drugs from the sea.

AU Gayler K.; Sandall D.; Greening D.; Keays D.; Polidano M.; Livett B.; Down J.; Satkunanathan N.; Khalil Z.
CS Australia. k.gayler@unimelb.edu.au
SO IEEE Engineering in Medicine and Biology Magazine, (2005) Vol. 24, No. 2, pp. 79-84. .
Refs: 43
ISSN: 0739-5175 CODEN: IEMBDE
CY United States
DT Journal; General Review
FS 030 Pharmacology
037 Drug Literature Index
LA English
ED Entered STN: 19 May 2005
Last Updated on STN: 19 May 2005
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L4 ANSWER 8 OF 12 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

AN 2004277714 EMBASE

TI One slip, and you're dead....

AU Nelson L.

SO Nature, (24 Jun 2004) Vol. 429, No. 6994, pp. 798-799. .

Refs: 8

ISSN: 0028-0836 CODEN: NATUAS

CY United Kingdom

DT Journal; (Short Survey)

FS 037 Drug Literature Index

052 Toxicology

LA English

SL English

ED Entered STN: 22 Jul 2004

Last Updated on STN: 22 Jul 2004

AB The lethal toxins produced by cone snails are in hot demand for neuroscience research, and are being developed as potent drugs. Laura Nelson visits a would-be snail 'farmer', for whom milking time is fraught with danger.

L4 ANSWER 9 OF 12 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

AN 2004146990 EMBASE

TI Conotoxins as selective inhibitors of neuronal ion channels, receptors and transporters.

AU Lewis R.J.

CS R.J. Lewis, Institute for Molecular Biosciences, University of Queensland, Brisbane, QLD 4072, Australia. r.lewis@imb.uq.edu.au

SO IUBMB Life, (2004) Vol. 56, No. 2, pp. 89-93. .

Refs: 20

ISSN: 1521-6543 CODEN: IULIF8

CY United States

DT Journal; General Review

FS 008 Neurology and Neurosurgery

029 Clinical Biochemistry

030 Pharmacology

037 Drug Literature Index

LA English

SL English

ED Entered STN: 15 Apr 2004

Last Updated on STN: 15 Apr 2004

AB Cone snails have evolved a vast array of peptide toxins for prey capture and defence. These peptides are directed against a wide variety of pharmacological targets, making them an invaluable source of ligands for studying the properties of these targets in normal and diseased states. A number of these peptides have shown efficacy in vivo, including inhibitors

of calcium channels, the norepinephrine transporter, nicotinic acetylcholine receptors, NMDA receptors and neurotensin receptors, with several having undergone pre-clinical or clinical development for the treatment of pain.

- L4 ANSWER 10 OF 12 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
AN 2003403019 EMBASE
TI Venoms to Drugs 2002 Conference: 14-19 July 2002, Heron Island, Queensland, Australia.
AU Craik D.
CS D. Craik, Institute for Molecular Bioscience, University of Queensland, Kalthera Pty. Ltd., Brisbane, QLD, Australia. d.craik@imb.uq.edu.au
SO IDrugs, (2002) Vol. 5, No. 9, pp. 881-884. .
ISSN: 1369-7056 CODEN: IDRUFN
CY United Kingdom
DT Journal; Conference Article
FS 008 Neurology and Neurosurgery
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 23 Oct 2003
Last Updated on STN: 23 Oct 2003
AB As the title suggests, the Venoms to Drugs conference was a highly focused meeting which reported on various aspects of venoms, with particular reference to the development of therapeutic agents from peptidic venom components. While the location on a coral island on the Great Barrier Reef reflected a focus on venoms from marine creatures, venoms from terrestrial animals and toxins from plants were also highlighted in a number of the presentations. Peptide components from the Conus marine snail species featured heavily in the program. Several talks referred to the progression through clinical trials of a least four known conopeptides. Regarding novel disclosures, Bruce Livett from the University of Melbourne gave a particularly interesting report on a newly discovered .alpha.-conotoxin with potential analgesic applications. This molecule is quite distinct from other conotoxins currently in clinical trials for the treatment of pain, and in particular from the .omega.-conotoxin class. .COPYRG. PharmaPress Ltd.
- L4 ANSWER 11 OF 12 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
AN 2001134193 EMBASE
TI Composition and therapeutic utility of conotoxins from genus Conus. Patent status 1996 - 2000.
AU Jones R.M.; Cartier G.E.; McIntosh J.M.; Bulaj G.; Farrar V.E.; Olivera B.M.
CS R.M. Jones, Cognetix Inc., 421 Wakara Way, Salt Lake City, UT 84108, United States. rjones@cognetix.com
SO Expert Opinion on Therapeutic Patents, (2001) Vol. 11, No. 4, pp. 603-623.
Refs: 51
ISSN: 1354-3776 CODEN: EOTPEG
CY United Kingdom
DT Journal; General Review
FS 008 Neurology and Neurosurgery
018 Cardiovascular Diseases and Cardiovascular Surgery
030 Pharmacology
032 Psychiatry
037 Drug Literature Index
039 Pharmacy

LA English
 SL English
 ED Entered STN: 30 Apr 2001
 Last Updated on STN: 30 Apr 2001
 AB With an exponentially increasing body of scientific evidence pointing toward the potential of conotoxins for treatment of a wide variety of nervous system and associated neurological disorders, there has been an explosion of activity in this patent area with more than eighty new patents and PCT publications in the past five years. With the emergence of ziconotide (SNX-111, .omega.-conotoxin MVIIA) as the first clinically used conotoxin for treatment of a neurological disorder, the first part of the new millennium is likely to see many more new filings in this field. The majority of the applications from this period focus on those classes of conopeptides that interact with nicotinic acetylcholine receptors (nAChRs) together with those that block voltage-gated ion channels. This arena has to date been dominated by three research groups: Neurex (a wholly-owned subsidiary of Elan, South San Francisco, CA, USA), Xenome and the Institute for Molecular Bioscience (IMB), University of Queensland (Melbourne, Australia) and Cognetix (Salt Lake City, UT, USA) together with the University of Utah Research Foundation and the Salk Institute for Biological Studies (La Jolla, CA, USA).

L4 ANSWER 12 OF 12 MEDLINE on STN
 AN 2006631497 MEDLINE
 DN PubMed ID: 17064074
 TI Cyclic MrIA: a stable and potent cyclic conotoxin with a novel topological fold that targets the norepinephrine transporter.
 AU Lovelace Erica S; Armishaw Christopher J; Colgrave Michelle L; Wahlstrom Maria E; Alewood Paul F; Daly Norelle L; Craik David J
 CS Institute for Molecular Bioscience, University of Queensland, Brisbane, Queensland 4072, Australia.
 SO Journal of medicinal chemistry, (2006 Nov 2) Vol. 49, No. 22, pp. 6561-8. Journal code: 9716531. ISSN: 0022-2623.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LA English
 FS Priority Journals
 EM 200612
 ED Entered STN: 27 Oct 2006
 Last Updated on STN: 19 Dec 2006
 Entered Medline: 5 Dec 2006
 AB Conotoxins, disulfide-rich peptides from the venom of cone snails, have created much excitement over recent years due to their potency and specificity for ion channels and their therapeutic potential. One recently identified conotoxin, MrIA, a 13-residue member of the chi-conotoxin family, inhibits the human norepinephrine transporter (NET) and has potential applications in the treatment of pain. In the current study, we show that the beta-hairpin structure of native MrIA is retained in a synthetic cyclic version, as is biological activity at the NET. Furthermore, the cyclic version has increased resistance to trypsin digestion relative to the native peptide, an intriguing result because the cleavage site for the trypsin is not close to the cyclization site. The use of peptides as drugs is generally hampered by susceptibility to proteolysis, and so, the increase in enzymatic stability against trypsin observed in the current study may be useful in improving the therapeutic potential of MrIA. Furthermore, the structure reported here for cyclic MrIA represents a new topology among a growing number of circular disulfide-rich peptides.